CASE REPORTS

Refer to: Gwinup G, Chelvam R, Jabola R, et al: Beer drinker's hyponatremia — Inappropriate concentration of the urine during ingestion of beer. Calif Med 116:78-81, Mar. 1972

Beer Drinker's Hyponatremia

Inappropriate Concentration of the Urine During Ingestion of Beer

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IN THE PAST SEVERAL YEARS we have seen a number of beer drinkers who have been admitted with striking hyponatremia, although we have been unable to find any reference to this condition in the literature. We report studies performed on such a patient in an attempt to establish the association between beer ingestion and hyponatremia and to gain information regarding the mechanism of the production of hyponatremia.

Report of a Case

The patient was a 46-year-old Caucasian man who was admitted to the Long Beach Veterans Administration Hospital 11 May 1970 with complaint of weakness and mild confusion. He gave a history of many years of drinking between six and twelve 16-ounce cans of beer a day. Consumption tended to be greater during the warmer summer months. For the preceding two weeks he had averaged eight to ten cans of beer daily. He denied vomiting or diarrhea and insisted that he maintained a reasonably normal dietary intake.

On examination of his hospital record it was found that he was admitted with similar but more severe complaints 15 July 1968, at which time his serum sodium content was 106 mEq per liter and again 8 June 1969, when serum sodium was 111 mEq per liter. On each of these occasions he was decidedly confused, but each time confusion abated spontaneously during the first several days in hospital without specific treatment. Past history revealed that for many years he had complained of selective weakness of the lower extremities, which had been ascribed to a peripheral neuropathy.

On physical examination he appeared well-nourished. The blood pressure was 150/90 mm of mercury and the pulse 78 per minute and regular. The neck veins were not distended and the thyroid gland was not enlarged. The chest was clear to auscultation and percussion, the heart of normal size and without abnormal sounds. The abdomen was soft, and there was no evidence of ascites. The liver was not enlarged. There was no pretibial edema. Muscular strength was essentially normal over most of the body but was symmetrically reduced in the lower extremities. Reflexes, sensation to pinprick, and proprioception were also diminished in the lower extremities.

On admission the hemoglobin was 14.1 grams per 100 ml, hematocrit 42 percent, white blood cell count 5,600 per cu mm with 62 percent polymorphonuclear cells and 38 percent lymphocytes. Specific gravity of the urine was 1.018 and it contained no protein, glucose, acetone or cells. Serum sodium was 122, chloride 84, potassium 5.1, and bicarbonate 24 mEq per liter, blood urea nitrogen 8 mg, cholesterol 232 mg, total protein 7.8 grams (albumin 3.3 grams) and fasting glucose 85 mg per 100 ml. SGOT was 33, alkaline phosphatase 9.5 King-Armstrong units, total bilirubin 0.8 mg per 100 ml, creatinine clearance 82

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ml per minute, *Bromsulphalein®* retention 4 percent at 45 minutes, prothrombin time 70 percent, protein-bound iodine 4.4 μg per 100 ml, and urinary 17 ketogenic steroids 8.9 and 17 ketosteroids 19.7 mg per 24 hours. X-ray films of the chest and the skull and an intravenous pyelogram were within normal limits. A brain scan was normal. Urinary porphyrins were normal.

No specific treatment or supplemental sodium was given, but within three days the patient's serum sodium had returned to 142 mEq per liter, his confusion had abated, and his strength was greatly improved.

Materials and Methods

Studies were conducted over a 35-day period while the patient was maintained on a general diet with no fluid or salt restriction. After a short baseline period, ten 16-ounce cans of beer containing alcohol 4.6 percent by volume and sodium 16 mg per liter were given over 16 hours of every day for seven days. This was followed by a control period of one week, after which ten 16-ounce beakers of water were taken over the same period of every day. After another control period, a quantity of alcohol equal to that contained in the beer was administered as 50 percent ethyl alcohol in seven doses of 2 ounces every two hours. Serum was obtained daily for determination of sodium, chloride, potassium, bicarbonate, and blood urea nitrogen. The volume and osmolality of each day's urine was measured, and the patient's weight and fluid intake were recorded daily.

Results

BUN ranged from 7 to 12 mg per 100 ml and showed no significant change throughout the study. During the period in which beer was administered, urinary sodium ranged between 33 and 47 mEq per 24 hours (mean = 42 mEq per 24 hours) with no significant change. At the beginning of the period of beer ingestion, the hemoglobin was 16.3 grams per 100 ml and the hematocrit was 47 percent. Ten days later, when the serum sodium had fallen to its lowest value, the hemoglobin was 14.3 grams and the hematocrit was 42 percent. At the end of the study the hemoglobin was 15.8 grams and the hematocrit 48 percent. At no time was the serum lipemic. The changes which occurred in serum electrolytes, body weight, and urinary osmolality are plotted in Chart 1 and the 24-hour total fluid intake and output volumes are recorded in Table 1.

During the period in which beer was administered, there was a progressive decrease in serum sodium, which had reached 123 mEq per liter on the last day of beer ingestion. Hyponatremia was accompanied by a comparable hypochloremia with minimal changes in serum potassium concentration. Changes in serum osmolality closely paralleled changes in serum sodium falling progressively from an initial value of 293 to 253 milliosmol per kilogram on the last day of beer ingestion. Body weight rose 8 pounds during the same period, and fluid balance was strongly positive, averaging almost 1,300 ml daily. Beer ingestion was accompanied by an inappropriately concentrated urine which remained above 800 milliosmol per kilogram throughout the last three days of the period.

Water ingestion produced an early decrease in both serum sodium and chloride concentration, but both returned to normal as the period continued. Body weight fell off slightly, and the urine became dilute averaging less than 300 mOs per kilogram. After the first two days, positive fluid balance was of far less magnitude than it was during the period of beer ingestion.

The ingestion of alcohol alone produced little or no change in serum electrolytes, body weight or fluid balance.

Discussion

In this patient the ingestion of large quantities of beer resulted in the production of concentrated urine, a pronounced increase in body weight, strongly positive fluid balance, and a rather striking degree of hypotonic hyponatremia which was presumably largely or wholly dilutional.

The combination of hypotonic serum and concentrated urine is often regarded as presumptive evidence for the inappropriate production of antidiuretic hormone. Although this state may occur as a consequence of bronchiogenic carcinoma and a number of other diseases, it has also been described in otherwise healthy persons, in whom it may be a self-limited condition.[1]

It is possible that in our patient ingestion of large quantities of beer served to unmask a tem-

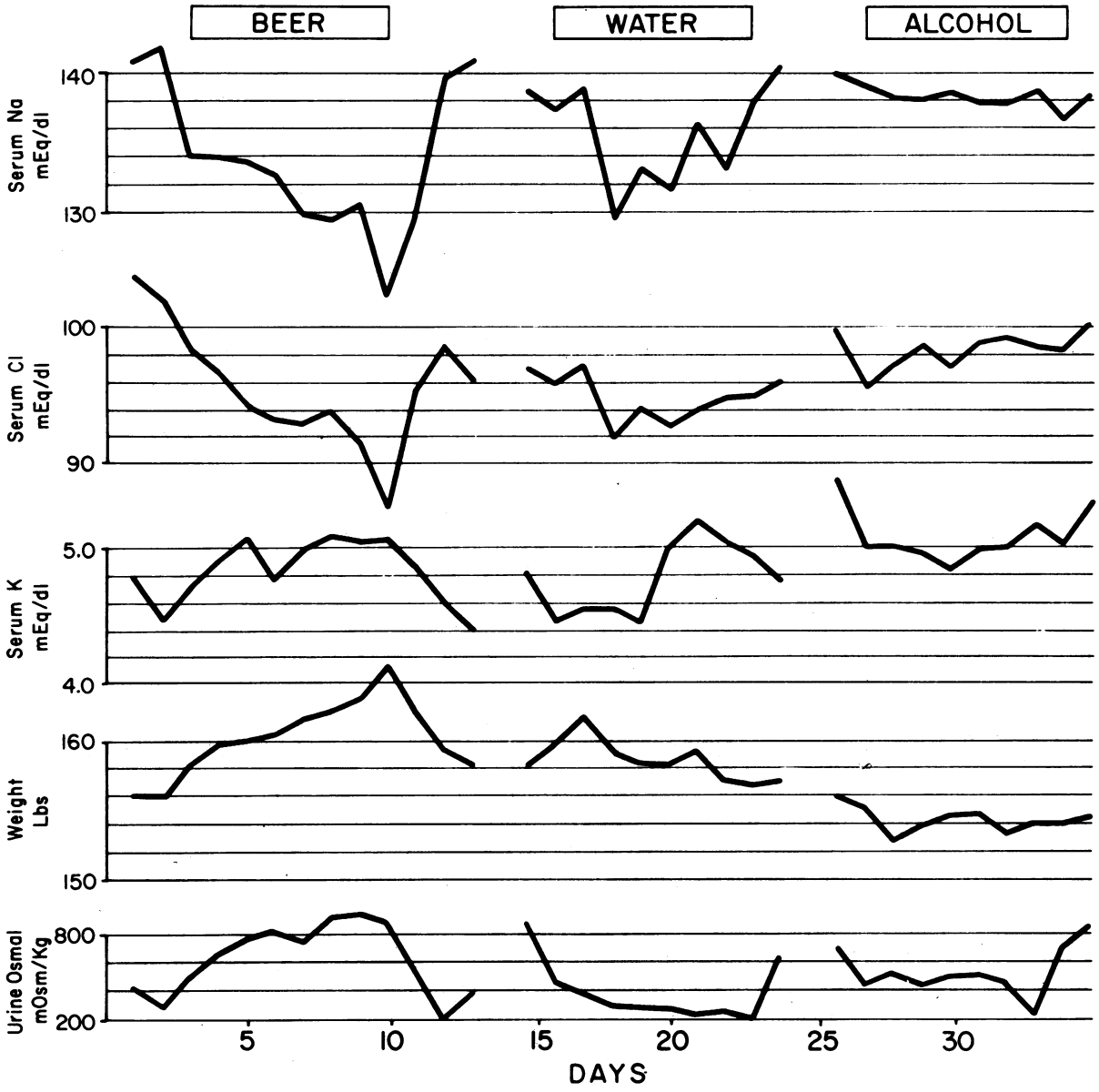

Chart 1.—Changes in serum electrolytes, body weight and urinary osmolality during periods of ingesting beer, water and alcohol

porary inability to dilute urine unrelated to any constituent of the beer except its water content. This explanation is supported by the unsustained decrease in serum sodium concentration in the first few days of the period of water ingestion. The hyponatremia of beer drinking might therefore be similar to the hyponatremia of the compulsive water drinker described by Hobson and English, in whom inappropriate antidiuretic hormone secretion was thought to play a permissive role.[2]

It is also possible that beer produced hyponatremia and inappropriate concentration of the urine by an unknown mechanism completely apart from inappropriate antidiuretic hormone secretion.

In our studies a fourth period was initiated in which 14 ounces of 50 percent ethyl alcohol and 160 ounces of water were administered daily, but the patient did not cooperate and this effort had to be abandoned. Additional studies will be necessary to determine the frequency of this syndrome and to further elucidate the mechanism by which it is produced.

TABLE 1.—*Daily Fluid Intake, Output and Balance Induced by Ingestion, Each for a Seven-Day Period, of Beer, Water and Alcohol*

	PERIOD		
	Intake (ml)	*Output (ml)*	*Balance (ml)*
BEER	5,810	4,120	+1,690
	5,400	4,160	+1,240
	5,930	5,100	+830
	5,800	4,680	+1,220
	5,690	4,220	+1,470
	5,480	4,410	+1,070
	5,910	4,100	+1,810
WATER	5,950	4,460	+1,490
	5,950	4,640	+1,310
	6,140	6,100	−20
	5,410	5,110	+300
	5,970	5,460	+510
	6,160	5,820	+340
	5,880	5,310	+490
ALCOHOL	2,100	1,910	+190
	1,960	1,440	+529
	1,680	1,210	+470
	1,400	1,640	−240
	2,010	1,770	+840
	1,640	1,240	+400
	1,500	1,180	+380

Summary

Studies were performed on a patient who had repeatedly presented to the hospital with profound hyponatremia following the consumption of large quantities of beer. The administration of 160 ounces of beer per day produced hyponatremia, weight gain, strongly positive fluid balance and inappropriate urinary concentration. It is concluded that in this patient the ingestion of large quantities of beer either unmasked or produced the syndrome of inappropriate concentration of the urine.

REFERENCES

1. Langgard H, Smith WO: Self-induced water intoxication without predisposing illness. N Engl J Med 266:378-81, 1962
2. Hobson JA, English JT: Self-induced water intoxication. Ann Intern Med 58:324-32, 1963

ADDENDUM: In the time between the preparation and the publication of this report Demanet et al* reported a similar group of patients with the same syndrome. All were heavy beer drinkers and presented with coma and striking hyponatremia.

JAMA printed an editorial on the subject in February of this year.†

*Demanet JC, Bonnyns M, Bleiberg H, et al: Coma due to water intoxication in beer drinkers. Lancet 2:1115-1117, 1971

†Water-intoxicated beer drinkers (Editorial). JAMA 219:1060, Feb 21, 1972

Refer to: Lewis JE, Sampson WI: PTC deficiency with phalangeal and interphalangeal (arthritic) changes. Calif Med 116:81-85, Mar 1972

PTC Deficiency with Phalangeal and Interphalangeal (Arthritic) Changes

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FACTOR IX (plasma thromboplastin component, Christmas factor) deficiency or hemophilia B, like its counterpart factor VIII (antihemophiliac globulin) deficiency or hemophilia A, is a well known cause of frequent and disabling episodes of bleeding into the larger joints of the body.[1,2,3] Bleeding occurs in approximately the following order of frequency: knees, elbows, ankles, shoulders, hips and wrists. Except for major bleeding episodes associated with specific traumatic events,[4,5,6] reports describing the changes that occur in the phalangeal and interphalangeal joints are rare.[1,2,4,7]

This communication presents the clinical history, physical examination, laboratory data, x-ray films and photographs of the changes which have occurred in the interphalangeal joints of a 29-year-old Caucasian man with severe PTC deficiency.

Report of a Case

The patient, a 29-year-old Caucasian man, had had bleeding at the site of circumcision shortly after birth. This led at that time to investigation at Stanford-Lane Hospital, San Francisco, where the diagnosis of severe plasma thromboplastin component (PTC) deficiency was established. Throughout early childhood he had numerous

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